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移動相の効果を含む感染症流行モデル

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1. Introduction

Transportation (i.e., population dispersal), a common phenomenon in human society, is considered as one of main factors that could cause the outbreak of some diseases such as influenza and SARS. It is reported that, in 2003, SARS broke out with some infection in an airplane: There was one person infected with SARS, and nine persons around the man were infected during the transportation. SARS broke out with such a kind of transport-related infection. A mathematical groundwork would be meaningful and useful in order to discuss such a transport-related infection. There have been many investigations concerning the effect of transportation (or population dispersal) on the spread of a disease (see [1, 2, 4, 5, 7–9, 11–13] and the references therein). However, few studies take account of the possibility for some individuals to become infective during transportation, and no paper discusses such a serious effect of transport-related infection in a more precise and strict way of theoretical/mathematical study about it. In this paper, we propose a multi-community model with an epidemic central place that can provide an reasonable and essentially simple idea of mathematical modeling to theoretically discuss the transport-related disease infection.

2. The model

The first step to model the transport-related infection is to use a disease transmission model based on the well-known patch models described by ordinary differential equations

with a geographically divided population (for the case of two patches, see [3]). However, in such modeling, the transport-related infection has been modeled as an instantaneous event, which is clearly an oversimplification or a mathematical convention.

Let us change the point of view for transport-related infection. We assume that a population is divided into the traveling phase where one travels and the non-traveling phase where one does not travel. Making use of the idea of compartmental modeling, we consider the mathematical model which is composed with a central place as the traveling phase and n communities as the community-specified non-traveling phase. This is an extended version of the phase-compartmental model in [6] and is formulated by the following $4n$ dimensional nonlinear differential equations:

$$
\begin{aligned}
S_i' &= B(N)S_i - \frac{\gamma S_i I_i}{S_i + I_i} + \mu I_i - \alpha_S^i S_i + \beta_S^i \tilde{S}_i, \\
I_i' &= \frac{\gamma S_i I_i}{S_i + I_i} - (\mu + D + \alpha_I^i) I_i + \beta_I^i \tilde{I}_i, \\
\tilde{S}_i' &= -\frac{\tilde{\gamma}\tilde{S}_i \sum_{k=1}^{n} \tilde{I}_k}{\sum_{k=1}^{n} (\tilde{S}_k + \tilde{I}_k)} + \alpha_S^i S_i - \beta_S^i \tilde{S}_i, \\
\tilde{I}_i' &= \frac{\tilde{\gamma}\tilde{S}_i \sum_{k=1}^{n} \tilde{I}_k}{\sum_{k=1}^{n} (\tilde{S}_k + \tilde{I}_k)} + \alpha_I^i I_i - \beta_I^i \tilde{I}_i, \qquad i = 1, 2, \cdots, n.
\end{aligned}
\tag{1}
$$

S_i and I_i represent susceptibles and infectives belonging to community i at the non-traveling phase, and $\tilde{S}_i$ and $\tilde{I}_i$ do those at the traveling phase. $\tilde{\gamma}$ is the infection rate at the traveling phase, and γ is that in every community at the non-traveling phase. These n communities are assumed to be identical except for the phase-transition rates between the community (non-traveling phase) and the traveling phase, α_s^i and β_s^i for susceptibles, and α_I^i and β_I^i for infectives.

This model can express more realistically the transport-related infection that traveling individuals are mixed at the traveling phase, and come back to their own community after the temporal traveling phase. You see that the traveling phase here plays a role of the central place, defined in the ecology, such that individuals from surrounding communities tensely interact there to each other. In the epidemic central place, that is, at the traveling phase, we assume no birth and no death since the time scale for the traveling is taken natural to be sufficiently smaller than that for the biological birth/death process in the

human case. Besides, we assume a population growth rate denoted by $B(N)$ for every community, where N is the total population size in the whole system. We set up the following basic assumptions about $B(N)$ for $N \in (0, \infty)$:

(A1) $B(N)$ is continuously differentiable with $B'(N) < 0$;

(A2) There is a $b > 0$ such that $B(b) = 0$;

(A3) $B(N) = B^+(N) - B^-(N)$ where B^+, B^- are nonnegative functions.

(A1) and (A2) involve the meaning of a density-dependent effect. (A3) is a technical assumption for deriving the basic reproduction ratio mentioned in the next section, while it has little restriction on our model in a biological sense (for example, in a logistic equation, B^+ corresponds to the intrinsic growth rate and B^- corresponds to the density-depentent effect). Furthermore, we consider the disease-related death rate D and the recovery rate μ at the non-traveling phase. We do not consider the recovery at the traveling phase (i.e., in the epidemic central place) because it is little likely that the infected person might recover during traveling.

3. Basic reproduction ratio

We now introduce the 'basic reproduction ratio' which is one of the most important key concepts in considering epidemiological models. In order to find the basic reproduction ratio of our model (1), we use a method established by [12], and lastly obtain the basic reproduction ratio R_0 for (1) as follows:

$$R_0 = \frac{\tilde{\gamma}\langle\Theta\rangle + n\gamma + \sqrt{(\tilde{\gamma}\langle\Theta\rangle + n\gamma)^2 - 4n\tilde{\gamma}\gamma\left(\langle\Theta\rangle - \frac{1}{n}\sum_{k=1}^{n}\frac{\alpha_I^k}{\beta_I^k}\right)}}{2(\mu + D)}, \tag{2}$$

where

$$\langle\Theta\rangle = \sum_{k=1}^{n}\left(\Theta_k \frac{\tilde{S}_k^*}{\sum_{k=1}^{n}\tilde{S}_k^*}\right)$$

with $\Theta_i = \frac{\alpha_I^i + \mu + D}{\beta_I^i}$, $i = 1, \cdots, n$, which we call the *infective transfer index*. Here $\tilde{S}_k^*$ $(k = 1, \cdots, n)$ are elements of disease free equlibria (DFE) E_0 given by

$$E_0 = \left(S_1^*, \cdots, S_n^*, 0, \cdots, 0, \tilde{S}_1^*, \cdots, \tilde{S}_n^*, 0, \cdots, 0\right)$$

with

$$\sum_{k=1}^{n}\left(S_k^* + \tilde{S}_k^*\right) = b, \qquad \alpha_S^i S_i^* = \beta_S^i \tilde{S}_i^*, \quad i = 1, \cdots, n.$$

As a result, R_0 is independent of any phase-transition rate of susceptibles, while it depends on the number of traveling susceptibles at the DFE. The dependence of R_0 on the infection rates γ and $\tilde{\gamma}$ is illustrated in Figure 1. The curve dividing the region into two areas for $R_0 > 1$ and for $R_0 < 1$ is given by

$$\gamma = \frac{\mu + D}{n} \times \frac{\tilde{\gamma}\langle\Theta\rangle - (\mu + D)}{\tilde{\gamma}\left(\langle\Theta\rangle - \frac{1}{n}\sum_{k=1}^{n}\frac{\alpha_I^k}{\beta_I^k}\right) - (\mu + D)},$$

which plays a role of the threshold for the disease spread.

4. Discussion

We successfully obtained the basic reproduction ratio as an explicit formulus of model parameters and the conventionally defined infective transfer index Θ_i, as shown in (2). Making use of the obtained basic reproduction ratio R_0, we can investigate how the disease invasion depends on the model structure. In order to have a further information about the disease invasion, let the infective transfer index be ordered as $\Theta_1 > \Theta_2 > \cdots > \Theta_n$ without loss of generality. Then, differentiating R_0 by $\tilde{S}_1^*$ and by S_n^*, we have

$$\begin{aligned}
\frac{\partial R_0}{\partial \tilde{S}_1^*} &= \frac{\tilde{\gamma}}{2(\mu + D)}\left(1 + \frac{\tilde{\gamma}\langle\Theta\rangle - n\gamma}{\sqrt{(\tilde{\gamma}\langle\Theta\rangle - n\gamma)^2 + 4\gamma\tilde{\gamma}\sum_{k=1}^{n}\frac{\alpha_I^k}{\beta_I^k}}}\right)\frac{\sum_{k=2}^{n}\tilde{S}_k^*(\Theta_1 - \Theta_k)}{\left(\sum_{k=1}^{n}\tilde{S}_k^*\right)^2} > 0 \\
\frac{\partial R_0}{\partial \tilde{S}_n^*} &= \frac{\tilde{\gamma}}{2(\mu + D)}\left(1 + \frac{\tilde{\gamma}\langle\Theta\rangle - n\gamma}{\sqrt{(\tilde{\gamma}\langle\Theta\rangle - n\gamma)^2 + 4\gamma\tilde{\gamma}\sum_{k=1}^{n}\frac{\alpha_I^k}{\beta_I^k}}}\right)\frac{\sum_{k=1}^{n-1}\tilde{S}_k^*(\Theta_n - \Theta_k)}{\left(\sum_{k=1}^{n}\tilde{S}_k^*\right)^2} < 0.
\end{aligned} \tag{3}$$

This result suggests that, if we decrease $\tilde{S}_i^*$ to suppress the number of traveling susceptibles of community i, the control may make the disease transmission situation worse due to the decrease in R_0 caused by it. Therefore, we can suggest that the public health control against a disease invasion would significantly depend on the nature of community structure including the connectivity between the member sub-commnities or the community-specified mobility of members in each sub-community. More detail discussion about our investigation from R_0 will be presented elsewhere.

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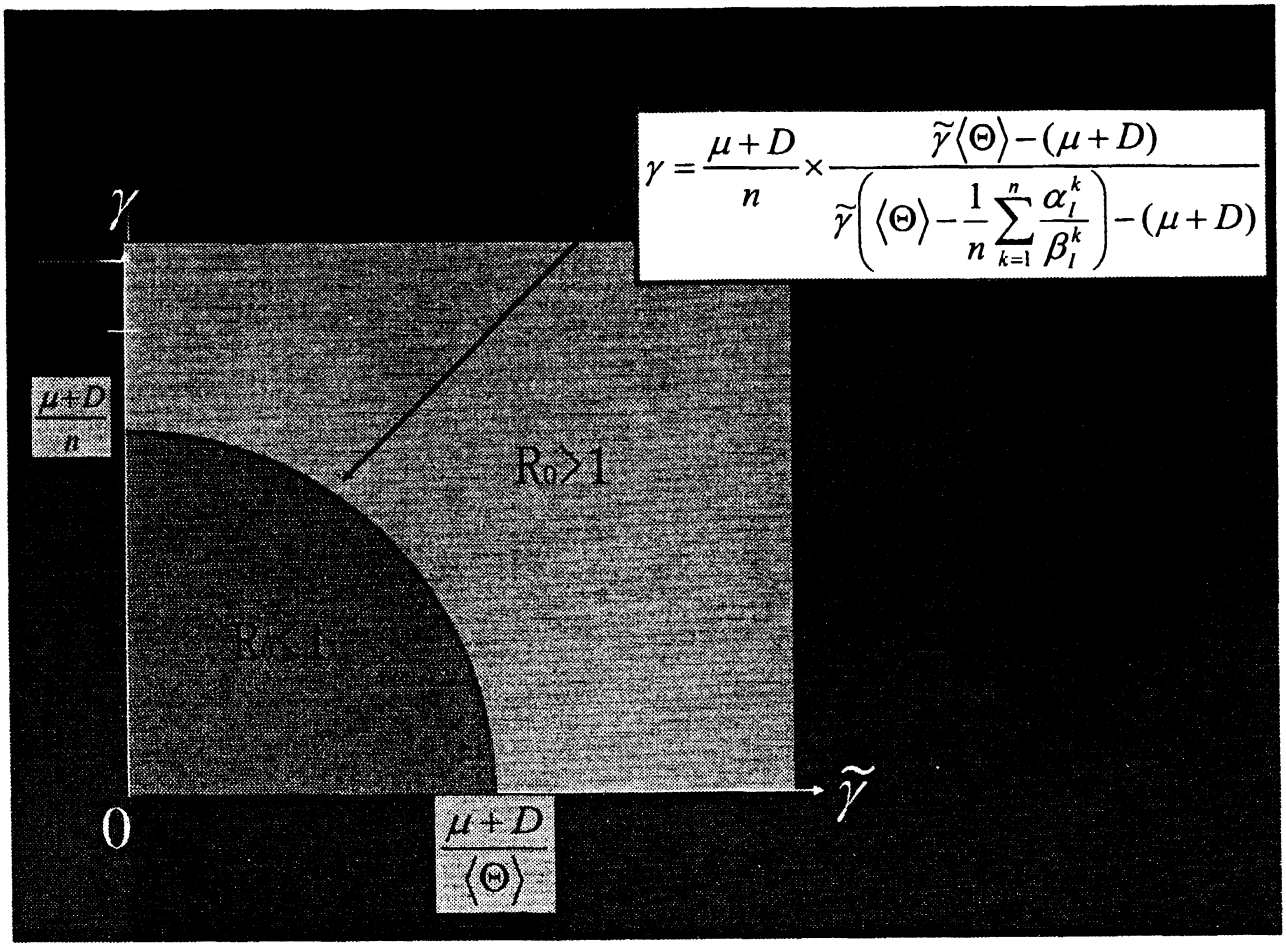

Figure 1. Illustration of R_0